

22

The First Heart Sound

JOEL M. FELNER

Definition

Heart sounds are discrete bursts of auditory vibrations of varying intensity (loudness), frequency (pitch), quality, and duration. The first heart sound (S_1) is composed of several high-frequency components; only the first two are normally audible. These two sets of audible vibrations are temporally related to closure of the mitral and tricuspid valves. The first heart sound coincides with contraction of the ventricles, thus identifying the onset of ventricular systole and the end of mechanical diastole.

Technique

The examination should be conducted in a warm, quiet room. Place the patient in a supine position after all clothing has been removed from the chest. Explain to the patient that you are going to examine the heart. Warm your hands and stethoscope, but warn the patient that your hands may be cool at first. The most comfortable and satisfactory position for most examiners is on the patient's right side. Since heart sounds may be palpable, attempt to palpate the first sound with the heel of the right hand and/or the fingerpads, initially at the cardiac apex and then over the entire precordium.

The acoustic events should be analyzed only after having assessed the venous, arterial, and chest wall pulsations. The auscultatory examination is commonly begun at the aortic area (second right intercostal space) and the stethoscope inched to the pulmonary area (second left intercostal space), the tricuspid area (lower left sternal border), and mitral area (cardiac apex), as shown in Figure 22.1. Follow an identical routine for every examination, passing from one part to another in a particular order.

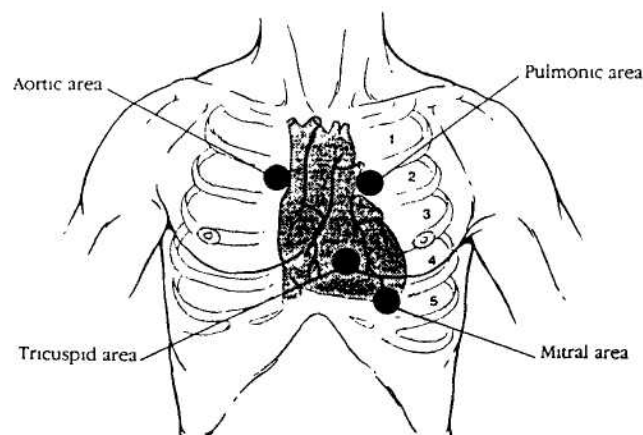


Figure 22.1
Routine areas for auscultation of the precordium.

Force yourself into the habit of listening for one sound at a time, using great care to time the heart sounds exactly. Listen with the stethoscope diaphragm, pressed firmly on the chest, to best hear the high-frequency vibrations of the first heart sound. Concentrate for several cycles on the quality and intensity of the first heart sound, dismissing everything else from your mind until the sound is clearly identified and appraised. The first sound is normally lower in pitch than the second sound.

The quality of the first heart sound (S_1) and its time relationship with the second heart sound (S_2) make it possible for the experienced observer to recognize a definite rhythm and thus easily distinguish between S_1 and S_2 . At normal and slow heart rates, S_1 is the first of the paired heart sounds, following the longer diastolic period and preceding the shorter systolic period. These sounds can be separated by simultaneously feeling or looking at the apex impulse while listening; the first heart sound is synchronous with the outward thrust of the apex impulse. With experience, it is possible to watch the movement of the stethoscope on the chest while listening to the heart sounds in order to time systole and diastole. When the apex impulse cannot be seen or felt, the pulsation of the carotid artery can be used as a guide. A finger on the carotid artery will sense the palpable arterial upstroke that immediately follows the first heart sound. With rapid heart rates, however, the slight delay between the first sound and carotid pulsation may make this maneuver unsatisfactory. Use of a more distant artery for this purpose leads to error because of the time it takes the pulse wave to reach the periphery.

For accurate identification of the first heart sound when multiple sounds are audible, move the stethoscope gradually from the second right to the fourth left intercostal space (ICS) by inching along the left sternal border. Then ask the patient to turn to the left lateral position. Examine the apical area while the patient is actually turning, using light pressure with the stethoscope bell. Other auscultatory areas that may be helpful in certain situations include the epigastrium and first or second left ICS. Auscultation should also be performed in the sitting position, especially in emphysematous patients, since the heart sounds may be distant or even absent in the supine position. Changing the position of the patient may accentuate sounds by bringing the heart closer to the chest wall or by accelerating blood flow due to exertion. The influence of respiration on sounds heard around the first heart sound should be noted. Also determine whether the first sound is split into its two components (mitral and tricuspid) and, if so, which component is loudest.

There is normal asynchrony in the closure of mitral (M_1) and tricuspid (T_1) valves, the mitral closure preceding tricuspid closure by 20 to 30 msec (0.02 to 0.03 sec.). This produces two audible components (M_1 - T_1) referred to as normal or physiologic splitting of the first heart sound. Such narrow splitting is usually best heard at the lower left sternal border with the stethoscope diaphragm. The tricuspid com-

ponent, which may increase with inspiration, is best heard in this location but is poorly transmitted to the apical region. The mitral component, in contrast, is best heard at the apex but is intense enough to be heard throughout the precordium.

Auscultation of heart sounds should not be performed as an isolated event. The first and second heart sounds are reference points for understanding the nature of certain pulsations. In order to correlate the physical findings and best understand cardiac physiology and anatomy, outline on a schematic diagram the heart sounds together with the jugular venous pulse, the carotid pulse, the precordial movements, and any murmurs and extra sounds heard, as shown in Figure 22.2. Careful study of this figure will illustrate many of the points of this section concerning timing, location, and intensity of normal heart sounds in relation to the other cardiovascular physical findings.

Basic Science

Figure 22.3 presents in graphic form the events of the cardiac cycle. The genesis of the first heart sound is contro-

versial. The recent use of echophonocardiographic and intracardiac techniques supports the concept, originally proposed by Leatham, that S_1 consists of two major elements temporally related to mitral and tricuspid closure. The actual contact of the valve cusps is not believed to be the source of sound. Therefore, the heart sounds likely represent vibrations of cardiac structures and blood within the heart. These vibrations are produced during the cardiac cycle by abrupt acceleration or deceleration of a mass of blood within the ventricles associated with sudden tensing of the entire atrioventricular (AV) valve apparatus (i.e., papillary muscles, chordae tendineae, valve leaflets, and rings) that stretches the surrounding structures to their elastic limits. The more rapid these forces, the louder the sounds and the higher the frequency.

At the onset of ventricular diastole, the mitral and tricuspid valves open, and their respective leaflets separate widely. Opening of these AV valves is normally silent. During ventricular filling, the leaflets of each AV valve begin to close; with the onset of atrial systole, the partially closed leaflets reopen. When atrial systole ends, the ventricle recoils and the leaflets close. Following closure, the AV valves are stretched toward the atrium by the momentum of the

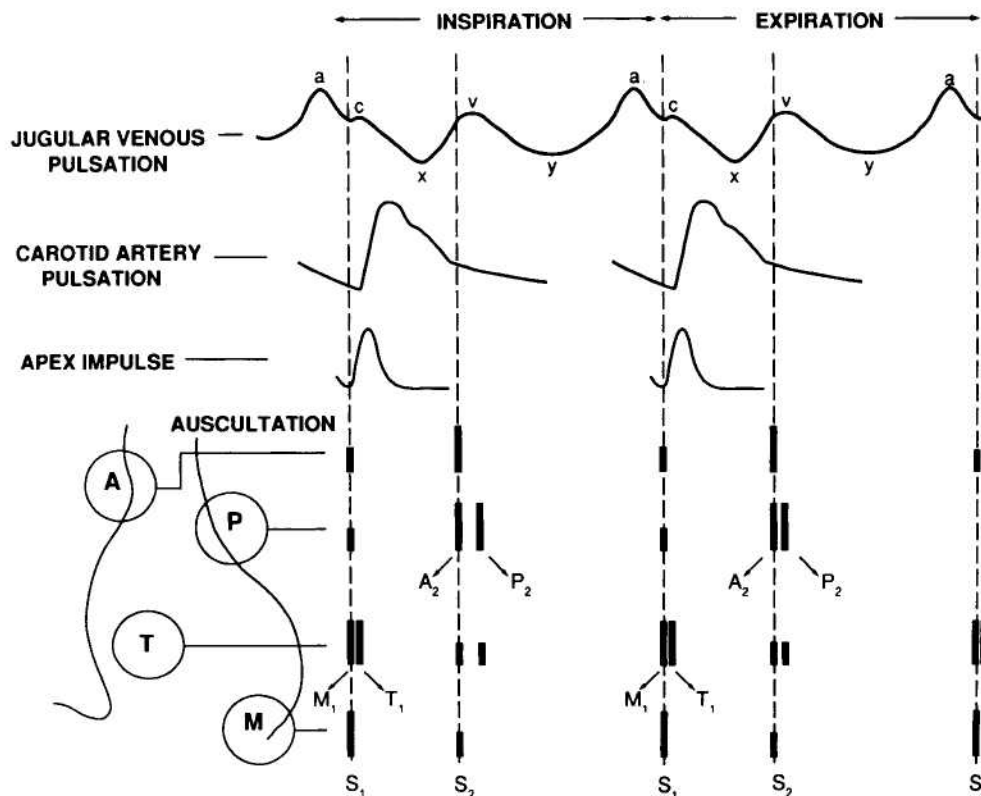


Figure 22.2

The normal cardiac pulsations and heart sounds. The *jugular venous pulsation* normally has 3 positive waves—a, c, and v—and 2 negative troughs—x and y. The “a” wave is approximately synchronous with the first heart sound (S_1) and just precedes the carotid upstroke. The “v” wave coincides approximately with the second heart sound (S_2). The normal *carotid artery pulsation* has a single positive wave during systole, followed by the diastolic notch (about the time of the second heart sound). The *apex impulse* represents the normal brief, palpable systolic impulse occurring at the time of S_1 . Auscultation at the aortic area reveals a normal S_1 and S_2 ; S_2 is normally louder than S_1 in this area. At the pulmonary area, there is normal inspiratory (physiologic) splitting of S_2 with the aortic component (A_2) preceding the pulmonary component (P_2). At the tricuspid area, there is normal splitting of the S_1 with the mitral component (M_1) preceding the tricuspid component (T_1). At this area, physiologic splitting of S_2 may also be appreciated. At the mitral area, S_1 is normally louder than S_2 , and only the aortic component is normally appreciated. Occasionally a third heart sound (S_3) is audible, reflecting deceleration of blood into the left ventricle during the rapid filling phase of early diastole. Children and young adults often have a normal (physiologic) S_3 .

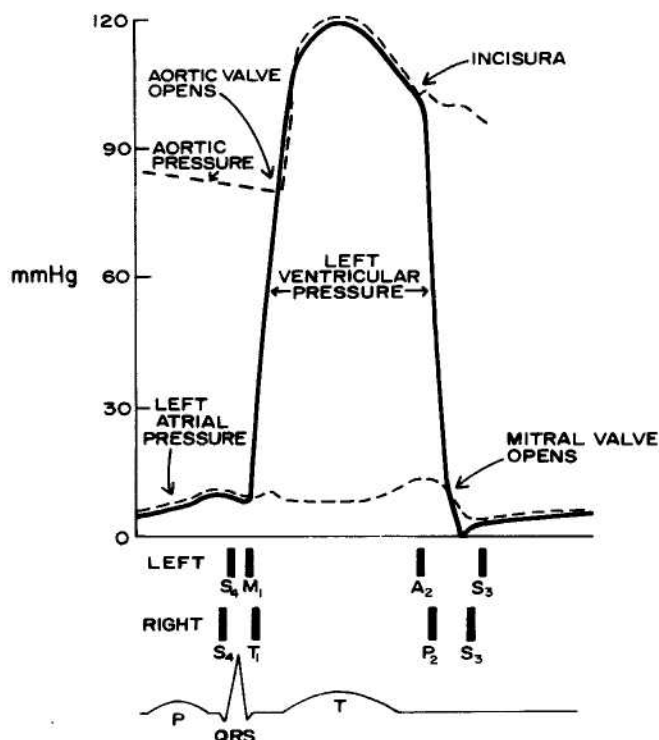


Figure 22.3

Events of the cardiac cycle. The first phase of ventricular systole, *isovolumic contraction*, begins with the abrupt rise in left ventricular pressure and is associated with the mitral component (M_1) of the first heart sound. The succeeding *rapid ventricular ejection* phase begins with the opening of the aortic valve and a rise in aortic pressure. This is followed by the phase of *reduced ejection* that ends just prior to the incisura on the aortic pressure tracing.

The onset of left ventricular *isovolumic relaxation*, which begins diastole, is usually identified with the incisura and the aortic component (A_2) of the second heart sound, whereas *rapid ventricular filling* begins when the left ventricular pressure falls below left atrial pressure. An S_3 may occur at the end of this phase. The subsequent phase of *slow ventricular filling* is followed by *atrial systole* during which an S_4 may occur.

Right-sided events include the tricuspid (T_1) and pulmonic (P_2) components of the first and second heart sound, respectively, and an S_3 and S_4 .

ventricular blood mass. When the AV valves reach their greatest excursion, the blood volume is abruptly checked (decelerated). These events set into motion a stretch-recoil sequence. The subsequent vibrations of this cardiohemodynamic system produce discrete sounds in the audible range that are heard on the chest wall as the first heart sound that is composed of the mitral closure sound (M_1) and the tricuspid closure sound (T_1).

The areas on the chest wall to which various acoustic events are preferentially transmitted include the second right ICS, the second and third left ICS, the fourth and fifth left ICS, the epigastrium, and the cardiac apex. Factors responsible for this transmission include the size and position of the heart in the thorax; the presence of fluid in, or fibrous thickening of, the pericardium; and the position and degree of aeration of the lungs.

Normal heart sounds will differ considerably in various chest wall locations and patient positions. In the region of the apex, for example, heart sounds are usually loud because the heart is in direct contact with the anterior wall of the thorax. In patients with thick chest walls or pulmonary

emphysema, heart sounds may be poorly heard or inaudible. They are heard more clearly if the patient bends forward or lies on the left side and is examined at the point of maximal expiration. In young persons with thin and elastic chests, heart sounds are heard with greater intensity than in older subjects whose chest walls are thicker and stiffer. If one or both lungs are retracted by disease, heart sounds over an area of the heart not covered by the lung will seem intensified. Therefore, before attributing abnormal heart sounds to disease of the heart, exclude such factors as these. In addition, several different patient positions (e.g., supine, left lateral, sitting, and, occasionally, prone) should be used during auscultation.

Cardiac auscultation is extremely difficult because the human auditory system is not well suited for the unusual vibratory characteristics of heart sounds and murmurs. Careful training, proper use of a good stethoscope, and concentration on selected portions of the cardiac cycle can help improve auscultation. A good stethoscope must have ear tips that fit snugly, a bend in the earpieces that aligns them properly with the ear canals, double tubes 25 to 30 cm (10 to 12 in) in length and 3 mm (.125 in) in internal diameter, a trumpet bell, and a diaphragm that attenuates low-frequency vibrations but does not alter high-frequency vibrations. Since auscultation is significantly influenced by background noise, the examination is best carried out in a quiet room.

Hearing the components of the first heart sound depends on the ability of the ear to integrate the frequency and intensity of the vibrations making up the sound. The ear can detect two sounds separated by an interval of as little as 0.02 second. A loud sound, however, may momentarily deafen the ear, with the result that an almost simultaneous faint sound may not be heard. For proper appreciation of the high-pitched components of the first sound, the diaphragm of the stethoscope should be applied with sufficient pressure to leave a mark (after-ring) on the chest when it is removed.

Clinical Significance

The first sound should be evaluated as to its quality, intensity, and degree of splitting. The normal first heart sound is audible in each of the four classic listening areas, and its intensity and quality will usually differ in each of these auscultatory areas. The first heart sound has a booming quality and is lower-pitched, duller, and longer than the second heart sound. It is usually louder at the apex than is the second sound. At the base, however, both components of the second sound are normally louder than the first sound (see Chapter 23, The Second Heart Sound). At the lower left sternal border, where the tricuspid closure sound (T_1) is best heard, splitting of S_1 is best evaluated.

The intensity (amplitude or loudness) of the first heart sound is affected by the position of the AV valve leaflets at end diastole (i.e., PQ interval of the electrocardiogram), and therefore their closing velocity, the force of ventricular contractility and the anatomical condition of the valves.

The intensity of the first sound is primarily related to the position of the AV valves at the onset of ventricular systole. The first sound is usually louder in subjects with a short PQ interval than in those with a long PQ interval. The shorter the PQ interval, the wider is the separation of the AV valve cusps when ventricular systole begins and the later is valve closure. This results in a more rapid valve-closing

motion and increased intensity of S_1 . Maximal intensity of S_1 occurs with PQ intervals of 80 to 120 msec (0.08 to 0.12 sec.). As the PQ interval progressively increases, there is a reduction in the intensity of S_1 . At PQ intervals greater than 200 msec (0.20 sec.) (first-degree heart block), there is less separation of the AV valves, which have already begun to close with atrial relaxation. Therefore, when ventricular systole begins, there is less excursion of the AV valves, and S_1 occurs earlier. Since less force is applied to the AV valves, their closing velocity is reduced, resulting in a softer S_1 . Although increased intensity of the first sound may be expected with shortening of the PQ interval, this does not apply to the short PQ interval of the Wolff-Parkinson-White syndrome, because the onset of ventricular contraction occurs later than is indicated by the onset of the electrocardiographic "delta wave."

A loud first heart sound is a hallmark of hemodynamically significant mitral stenosis. Mobile but stiff mitral leaflets produce a loud S_1 unless the leaflets are heavily calcified. The loud S_1 is due primarily to greater excursion of the leaflets during closure, since elevated left atrial pressure has kept the leaflets relatively wide apart. In addition, stiff, noncompliant leaflets and chordae tendineae appear to resonate with increased amplitude. A similar mechanism is responsible for the loud S_1 in patients with left atrial myxoma.

Conditions that increase myocardial contractility, including hyperadrenergic states (e.g., exercise, anxiety, anemia, fever, pregnancy, and thyrotoxicosis), also tend to be associated with a loud S_1 . The prime factor in increasing the intensity of S_1 in these conditions is the increased rate of pressure development in the ventricles. The first sound also tends to be louder in young people and in patients with thin chest walls.

Decreased intensity of S_1 occurs in conditions that cause the AV valves to close prior to ventricular systole or if there is a reduction in the rate of intraventricular pressure development. First-degree heart block is the most common cause of a soft S_1 . Holosystolic mitral regurgitation, ventricular septal defect, and acute aortic regurgitation also reduce the intensity of S_1 . In both holosystolic regurgitation and ventricular septal defect, the intensity of S_1 may be decreased or masked by the murmur itself, the isovolumic period may be absent, or the rate of rise of intraventricular pressure may be blunted. In acute severe aortic regurgitation, S_1 is most commonly diminished because preclosure of the mitral valve occurs as a result of the rapid increase in left ventricular filling pressure.

A decrease in the intensity of S_1 is also associated with myocardial depression (e.g., cardiomyopathy, myxedema, acute myocardial infarction, and cardiogenic shock) because the rate of intraventricular pressure development is reduced. Pulmonary emphysema, because of the increased amount of lung tissue interposed between the chest wall and the heart, also tends to attenuate S_1 .

Beat-to-beat variations in the intensity of S_1 occur in conditions that vary the PQ interval (e.g., second-degree AV block, Wenckebach type), the presence of AV dissociation (e.g., complete heart block and junctional or ventricular tachycardia), or during variable rates of intraventricular pressure development (e.g., atrial fibrillation and pulsus alternans). S_1 intensity may also vary in electrical alternans.

Splitting of the first heart sound into its two audible components, M_1 and T_1 , is a normal finding on cardiac auscultation. The M_1 - T_1 interval is normally separated by

20 to 30 msec. The fact that the first heart sound is split may be helpful in certain disease states. For instance, a loud tricuspid component of the first heart sound may be heard in patients with Ebstein's anomaly, right atrial myxoma, atrial septal defect, and the straight-back syndrome. The mitral component of the first heart sound is extremely loud and may be heard throughout the precordium in patients with mitral stenosis.

Wide splitting of the first sound is almost always abnormal. The split may be increased to 60 msec in patients with right bundle branch block, Ebstein's anomaly, or other conditions in which there is electrical delay in activation of one of the two ventricles (e.g., ventricular ectopic beats, ventricular tachycardia, AV block with idioventricular rhythm, and left ventricular pacing). Splitting of the first sound is not characteristic of left bundle branch block because there is no significant delay in onset of left ventricular contraction. Mechanical delays in closure of the mitral valve (e.g., mitral stenosis and left atrial myxoma) or the tricuspid valve (e.g., right atrial myxoma) may cause abnormal splitting of the first heart sound.

Sounds occurring around the time of S_1 that must be differentiated from a split first sound include an atrial gallop or fourth heart sound (S_4), an ejection sound (ES), and an early systolic click. The fourth heart sound, which forms the S_4 - S_1 complex, is a low-pitched, late diastolic (presystolic) sound. The S_4 may emanate from either the left or right ventricle. When left-sided, the much more common occurrence, it is confined to the cardiac apex where it is associated with palpable presystolic distention of the apex impulse. It is best heard with the bell of the stethoscope placed lightly over the apex, barely making an air seal, and is attenuated with increased pressure on the bell (i.e., creation of a diaphragm). It is usually reduced in intensity with standing (i.e., reduced venous return).

The ejection sound that forms the S_1 -ES complex is a high-pitched, early systolic sound. It may be heard at the base where splitting of S_1 is not heard or at the cardiac apex if it emanates from the aorta or aortic valve. There is no reliable maneuver that will differentiate these two acoustical events. The systolic click of mitral valve prolapse, in contrast, occurs later in systole than the ejection sound, and varies in location in systole with certain maneuvers that change the shape of the left ventricle.

References

- Craige E. Echocardiography in studies of the genesis of heart sounds and murmurs. In: Yu P, Goodwin J, eds. *Progress in cardiology*, vol 4. Philadelphia: Lea and Febiger, 1975.
- Leatham A. The first and second heart sounds. In: Hurst JW, Logue RB, Schlant RC, Wenger NK, eds. *The heart*, 6th ed. New York: McGraw-Hill, 1985.
- Levine SA, Harvey WP. *Clinical auscultation of the heart*. Philadelphia, W. B. Saunders, 1959;1-97.
- Luisada AA. *The sounds of the normal heart*. St Louis: Warren H. Green, 1972.
- Luisada AA. *The sounds of the diseased heart*. St Louis: Warren H. Green, 1973.
- Shah PM. Hemodynamic determinants of the first heart sound. In: Leon DF, Shaver JA, eds. *Physiologic principles of heart sounds and murmurs*. New York: American Heart Association, 1975;2-7.

Stein PD. A physical and physiological basis for the interpretation of cardiac auscultation. New York: Futura, 1981.

Tavel ME. Clinical phonocardiography and external pulse recording, 2d ed. Chicago: Year Book Medical Publishers, 1972;35–38.

Thompson ME, Shaver JA, Leon DF, et al. Pathodynamics of the

first heart sound. In: Physiologic principles of heart sounds and murmurs. New York: American Heart Association, 1975;8–18.

(See also references for The Second Heart Sound, Chapter 23.)